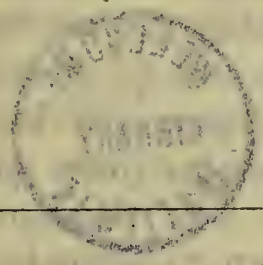
ON THE

ANATOMY OF PNEUMONIA.

By WILLIAM HENDERSON, M.D., Edin.,

PHYSICIAN TO THE ROYAL INFIRMARY OF EDINBURGH, AND LECTURER ON PATHOLOGY AND PRACTICE OF MEDICINE.



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EDINBURGH: BALFOUR AND JACK.

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The effects of inflammation on the condition of the lungs in common acute pneumonia have been, in respect to some particulars, stated so differently by pathologists of the highest eminence, that it appeared to me that there was a necessity for a more minute examination of the anatomy of the disease than had hitherto been made. And not only as to certain points of difference among authors on the subject, did it seem desirable to make more minute investigations; but also as to some particulars on which their researches have been confessedly defective, and as to others which have been adopted on grounds which are obviously inadequate. Several of the most important points, at present the subjects of disagreement among pathologists, will be found in the following pages illustrated by the assistance of the microscope; and I may remark of the results which have been thus obtained, that they are very easily established, after a little practice in the use of the instrument, and are not deductions from an over-nice distinction of obscure or inconstant objects.

There is less discrepancy of opinion, respecting the characters and nature of the first striking change produced in the lungs by inflammation, than respecting those of the subsequent stages. An excess of blood, an effusion of serous fluid, and a decrease in the firmness of the tissue, are the familiar signs of this state. But as these conditions have been generally admitted as the not uncommon results of a passive congestion of the lungs, as well as of inflammation, almost every recent author who has treated of pneumonia, maintains that there is no anatomical ground for distinguishing the engorgement of inflammation in its first degree, from that of passive congestion. Andral, after reviewing the anatomical characters of pneumonia in its first stage, and of passive congestion of the lungs, concludes in the following terms:—

"Now, as the anatomical characters of the hypercœmia are precisely the same in both these cases, it follows that, in this instance at least, the true nature of the morbid lesions found on dissection, can only be known by the nature of the symptoms du-

through the bronchial tubes. The third was a case in which death had occurred after fifteen or sixteen hours of very imperfect respiration, in consequence of cerebral disease. The exact nature of the case having been obscure, and suspicion having been entertained of poisoning by morphia, the respiration had been maintained for some hours artificially after the natural efforts had become rare and imperfect; but for a long time before death the patient had been left to breathe unassisted, and very insufficiently. The lungs were loaded so much with dark blood, and rendered so dense, especially at the posterior and lower parts, that a portion, when detached and thrown into water, sank immediately to the bottom of the vessel. From the previous examinations of inflamed lungs which I had made, and from my not having had opportunities of examining any specimens of passive engorgement so remarkable as those I have described, I felt much interest in them—the more especially that, in addition to the sero-sanguineous engorgement, the firmness of the pulmonary tissue was considerably lessened in two of them, and in the third appeared to be retained in consequence of previous inflammatory attacks, which had left numerous tough cellular bands intermingled with the substance of the organ. I therefore inspected them with much care, and subjected the fluid which was contained in them to the strictest scrutiny, but could detect no trace of the bodies to which I refer. They are not, however, peculiar to common or simple inflammation, for I have seen them in great abundance in the acute tubercular infiltration of the lungs, while the infiltrated matter had still but little consistence.

From the considerable size which these bodies possess, they assist in imparting a particular appearance to the fluid of pneumonia, when viewed even with the naked eye, which is not distinguishable in that of passive infiltration. The pneumonic fluid is commonly noticed as merely a bloody serum, and frequently the sanguineous tinge conceals the other character it possesses. But if care be taken to prevent its acquiring much of this colour by admixture with the blood of the divided vessels of a section of the lungs, by washing the surface well before the expressed fluid is examined, it will be found to present a certain degree of greyish turbidness, dependent chiefly on the existence of minute whitish specks. These are easily ascertained to be the bodies in question. If the pneumonia have existed in the posterior and lower parts of the lungs, and if much blood be still present in the tissue, the bloody colour given to the fluid by the escape of the colouring matter from the vessels after death, will prevent the above peculiarity of the pneumonic fluid from being seen. But in other circumstances, it is capable of being detected with ease, as the expressed fluid issues in a stream from the divided bronchi; for this fluid is contained chiefly in the air cells, as may be de-

termined by attentively regarding the surface from which it is made to exude by pressure, when it will be perceived, that at particular points, here and there, the exudation is much more abundant than elsewhere, and flows from minute orifices. That these belong to the bronchial tubes is rendered plain by the occasional appearance of air bells in the current that flows from them, as well as by the constitution of the fluid itself, which I shall notice hereafter. The mere circumstance of the fluid escaping in this way is not, however, peculiar to pneumonia. It may be observed also in infiltration from passive engorgement. By some it is supposed that the large quantity of thin serous fluid presented after death in lungs or parts of the lungs, in the first stage of inflammation, is not due to secretion. Dr Williams, in his excellent treatise on Diseases of the Chest, says of it:—"As there is no serous expectoration in the first stage of pneumonia, I cannot but regard the serum found in the lung after death, as in great measure the result of the cadaveric separation of the serum from the blood in the engorged vessels;" (p. 133) and in the preceding page, he expresses his belief, that at this stage the small air cells contain nothing but the "characteristic viscid secretion of the mucous membrane." The following considerations will, I think, shew that these opinions are incorrect. In the first place, as viscid mucus does not dissolve in water, it is impossible that the fluid of pneumonic engorgement could present the appearance which it does under the microscope. When viewed through that instrument, the characteristic secretion of the mucous membrane, the mucous globules, or cells of the epithelium, are found to be swimming freely and separately in the thin fluid, and are not stationary, as when suspended in a viscid medium. On the supposition that they had been secreted along with a viscid substance possessing the properties of mucus, they could not present the appearance I have mentioned, because a watery fluid could not have disengaged them from the insoluble and glutinous matter in which they were contained. In the second place, the fluid, thin and watery, as it has been described, may sometimes be found in a part of an inflamed lung which has lost its sanguineous colour, and, consequently, contained no blood to yield the fluid by cadaveric separation. Of this fact, I had an illustration very lately. The inflammation had affected, along with other parts, the anterior of the apex of the right lung. This part had, for about an inch and a half from the highest point, a considerable degree of density to the touch, and was of a pale stone colour, with a slight dash of pink. On pressing, there issued a copious flow of very thin and slightly turbid serosity, almost entirely from separate and distinct orifices, and exhibiting a very few air bells. Nothing can be fairly argued from the nature of the expectoration, as to the kind of fluid in the air cells, for we not unfrequently find

that there is no expectoration at all, even when auscultation proves that there is much fluid in the cells,—a fact which shows that there is no necessary connection between expectoration and the fluid in question.

The presence of the granular body which I have mentioned, is not the only important peculiarity in the serous fluid of the inflamed air cells. The cells of the epithelium undergo more or less generally, a decided alteration, changing their common oblong, or oval, into a round figure, decreasing in size, and losing much of their transparency. These alterations have been noticed by Vogel, as the first effects of inflammation on the secretion of the bronchial mucous membrane, preparatory to the appearance of pus globules, which do not present themselves at so early a period. When the inflammation is very recent, the fluid contains some healthy epethelium cells, mingled with those which have undergone the inflammatory changes. I shall have occasion to allude to these changes more particularly in treating of the subsequent stages of pneumonia; but I may remark in this place, that I have examined them so repeatedly in pneumonia, and had previously so often done so in the expectoration of various degrees of bronchial inflammation, that I have not the smallest doubt of the reality of the alterations produced in the cells of the epithelium by inflammation, and of their importance as a sign of the tissue having been inflamed, in which they are found. In the three cases of extreme sanguineous engorgement and serous infiltration of the lungs to which I have already referred, the cells of the epithelium, which floated in the contents of the air cells, were large, transparent, and oval, as in the natural state. How they were separated from the mucous membrane does not appear, but they existed in considerable abundance, mingled with blood-discs, which had made their way into the cavity of the cells. The foregoing observations lead to the conclusion, that the mucous membrane of the air cells is the proper seat of the inflammation in pneumonia, but it may be also shown that the morbid action is not strictly confined to the cells, but extends also to the intervesicular tissue, for an effusion of fluid into this tissue can be demonstrated by the following simple process. Let the pleura, and the subjacent fibrous layer which invests the pulmonary lobules, be carefully stripped, or dissected off from the surface of a recently inflamed portion, and then, by gentle pressure, a small quantity of serous fluid may be disengaged from the cellular tissue, and may be seen passing out between the air cells, which, if the operation be properly conducted, will appear amidst the exuding moisture, distended with air, forced into them by the pressure on other parts.

The chief disagreement among those who have written on the

pathology of pneumonia, exists in regard to the cause of the granulated appearance which is presented at a more advanced period of the inflammation than that to which I have hitherto referred. I notice first the opinion of Laennec which is expressed in the following terms, when he treats of the granulations of the second degree of pulmonary inflammation—that to which the designation of red hepatisation has been given. "These are evidently the air cells converted into solid grains by the thickening of their parietes, and the obliteration of their cavities by a concrete fluid."[1] This view had been previously expressed more at large in his Clinique Médicale, by Andral, who had founded his conception of the tissue actually inflamed in pneumonia on the crepetant rattle, which is present when the inflammatory secretion is agitated by air, and which from its minuteness he could not but refer to the finest part of the apparatus, as the larger rattles pertain to those which afford a larger space for the mutual action of the air and fluid. "If this proposition be exact," he adds, "it follows that pneumonia consists essentially in inflammation of the pulmonary vesicles, the internal surface of which secretes a liquid at first muco-sanguineous, then purulent. In proportion as the inflammation advances, the liquid secreted becomes more thick and viscid; it cannot now be expelled from the cavity where it is formed; it accumlates there, obstructs and distends it, and gives origin to the numerous granulations of which the tissue of the lung, in red hepatisation, appears specially formed."[2] This opinion, which found acceptance with Laennec, has been also adopted by Louis, who endeavoured to illustrate its accuracy by the experiment of injecting the air cells by the bronchial tubes, by which he says, if the injection be pushed gently, the lungs are made to present an infinity of little masses, which, on being divided, offer precisely the granulated aspect of the second degree of pneumonia.[3] More recently Andral appears to have become dissatisfied with this explanation of the nature of the granulations, and in the following passage gives a different view of their constitution:—"The morbid alteration known by the name of hepatisation of the lung, is produced by a considerable degree of sanguineous congestion of the parietes of the capillary bronchia, and air cells, the effect of which is to diminish or obliterate their cavities."[4] This is also the opinion of Hourmann and Dechambre, who ascribe the granulated appearance to a kind of erection of the walls of the air cells, by which their cavity is obliterated, and their permeability destroyed.[5] Dr

[1] On Diseases of the Chest, Forbes' Translation, p. 201.
[2] Clin. Médicale, t. iii, (1824,) pp. 312, 313.
[3] Recherches Anat. Path. sur la Phthisie, (1825,) p. 9.
[4] Path. Anatomy, (Translation,) (1831,) p. 512.
[5] Arch. Générales, t. x, 1836.

Stokes, too, coincides with the view of Andral. His words are, —"We must agree with Andral in the opinion that the solidity of acute pneumonia arises not from any deposition of lymph, but merely from an excessive congestion of blood."[1] Dr Williams' opinion differs from both of those which I have mentioned. "Many minute examinations," says he, "which we have made of hepatised lungs, have convinced us that the granulations contain no viscid mucus."[2] "They appear rather to consist simply of the little bunches of vesicles, whose membraneous tunics have been so swelled by the deposition of a soft albuminous matter in them (the tunics,) as well as from the increased size of their blood vessels, that their cavities are obliterated."[3] And, again, "this change consists in the gradual effusion of a semi-solid albumen in the interstitial tissues, which, pressing on and obliterating the cavities of air cells and smaller bronchi, destroys the spongy texture of the lung, and converts it into a more or less solid mass."[4] Professor Rokitanski of Vienna, in his excellent work on pathological anatomy, now in process of publication, has reverted to the original opinion of Andral, which he re-asserts in much the same terms as those which I have quoted from the Clinique Médicale.

By the same methods of investigation as those which I have already mentioned, I have followed the successive changes of the more advanced conditions of the disease, and with so much facility and exactness, as to admit of my giving the following particulars of the pulmonary granulations. When one or two of the red granulations are cut out, and bruised on a plate of glass, they are found to yield a thickish fluid. When this is submitted to examination by the microscope, it is found to contain, along with some blood-discs and small granules, numerous globular bodies, of a considerably larger size than blood-discs or pus globules. They correspond exactly with the description and figures which Vogel gives of the cells of the epithelium thrown off from an inflamed mucous surface, and with the globules which abound in the expectoration of recent acute catarrhs, and in old catarrhal expectoration which is not of a yellow colour. Their presence in the secretion of the air-cells in the granulations of pneumonia, proves that the epithelium extends into these minute recesses, and the alterations which they undergo, corresponding as they do with those which inflammation effects in the epithelium of the bronchial tubes, prove the existence of inflammation in the tissue from which they are detached. In reference to these alterations, Vogel remarks, "If the mucous membrane become inflamed, the

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[1] A Treatise on the Diagnosis and Treatment of Diseases of the Chest, part i, p. 312.
[2] Cyclop. of Practical Med., t. iii, p. 410.
[3] Loc. cit.
[4] On Diseases of the Chest, (1840,) p. 134.

altered mucus no longer exhibits normal epithelium cells,—they have undergone a pathological change. They become smaller, from $\frac{1}{80}'''$ to $\frac{1}{100}'''$ in diameter, (the larger ones in the healthy state being from $\frac{1}{30}'''$ to $\frac{1}{60}'''$ long, and about $\frac{1}{60}'''$ or $\frac{1}{80}'''$ broad,) and more rounded; the outer membrane appears denser, more sprinkled with granules, and less transparent than normal mucous cells; the dimmer and more opaque the outer membrane becomes, the less clearly does the nucleus appear, and sometimes it can no longer be seen,—sometimes in its place two or three smaller nuclei."[1] The healthy cells of the epithelium are so transparent and delicate in their outline, that where a difference of size or figure may not be so striking as to distinguish them in every instance very decidedly from the cells of the inflamed membrane, the degree of opacity of the latter, and especially their strongly defined margin, present such decided characters of difference, that they cannot be confounded. (See figs. 2 and 3.)

The blood-discs which are observed in the debris of the red granulations, would appear to be lodged in the vessels of the air-cells, for as the secretion accumulates, and the red tinge of the cells disappears, not a trace of them is to be found. The red colour of the granulations gives place gradually to a pale pink, this to a pale straw colour, in some instances of a waxy lustre, when viewed through a lens, and this to an opaque drab colour, which passes, in extreme cases, into yellow. Granulations possessing these various tints may be seen sometimes on the surface of the same section, blending gradually in the succession I have mentioned. The reddish colour of the granulations disappears first from their summits, and may be traced before finally ceasing, in the intervesicular spaces, in the form of narrow lines. Ultimately these disappear, and with them, seemingly, the very existence of intervesicular spaces; for the large granulations of the pale kind appear to be in contact at their bases. Pus globules, (fig. 4,) in very small quantity, may sometimes be noticed in the bruised granulations before the red colour is lost, and they increase in number the nearer the colour of the granulations tends to yellow. Still they are comparatively few in the pale straw-coloured granulations, of which the grey hepatisation is the most commonly composed. This colour seems to depend chiefly on the dim epithelium cells, and on the consistent matter in which they are suspended. The common grey hepatisation, therefore, is not so much a stage of true suppuration, as of infiltration with mucus, the cells of which have not yet advanced into the purulent condition, as Vogel supposes them to do in the ultimate stage of their mutation, when the inflammation persists unabated, or increases

[1] Physiologisch-Pathologische Untersuchungen über Eiter, &c. Von Julius Vogel. (Erlangen 1838,) p. 149.

in degree. The ascertaining of this point has satisfied me on a subject which previously excited considerable surprise. I have repeatedly had occasion to examine the lungs of persons, more especially of children, who had died after having laboured under the condensation consequent on pneumonia, for several months, in which the granulations presented generally more or less of the pale straw-colour, while yet the tissue retained a very considerable degree of firmness—a circumstance which appeared remarkable on the common supposition that the colour in question was due to the accumulation of pus, which is so constantly associated with much softening of the texture in which it exists. Another reflection, also, of some interest, occurred to me on considering the close grouping of the distended cells after the red colour had disappeared. The cells being confined in their separate lobules by a firm fibrous envelope, it appeared probable that the accumulation of a secretion in their interior, too viscid to escape easily by the minute bronchial twigs, might have the effect of compressing the blood-vessels of the cellules, and of the intervesicular tissue, and so of obliterating them, in consequence of the counter-pressure of the interlobular septa. I have not had an opportunity of ascertaining the fact, by attempts to inject a portion of hepatised lung; but in the case of rapid and extensive tubercular infiltration of the cells, which is essentially analogous in the particular in question, the obliteration or compression of the blood-vessels is very remarkably illustrated by artificial injection. I have several specimens of the latter disease in the yellow state, in which the blood-vessels injected in the common way, are very sparingly demonstrable in the infiltrated portions, and those which are so, are almost entirely the more considerable branches. If such be the effect of distention of the cells with consistent mucus, it may be supposed to exert a salutary influence on the inflammation; and on this ground we may cease to be surprised at those instances of almost complete freedom from suffering or constitutional disturbance, which occur not very seldom, though a large portion of the lungs is in a state of pneumonic condensation.

Those who think that the red granulations are the result of distention, or turgescence of the parietes of the air-cells, refer the alterations of colour which I have described to changes occurring in the same situation. Andral appears to regard the grey colour as dependant on the removal of the colouring matter of the blood, which is contained in the distended vessels of the cells, and throws out a suggestion, that what is termed the purulent infiltration may be regarded in the same light—the difference being—that the blood has not only lost its colouring matter, but its ordinary degree of consistence. Dr Williams thinks the change of colour which precedes suppuration, owing to the "sub-

stitution of more of the yellowish-white semi-solid albumen for the red particles"[1] in the walls of the cells. Hourmann and Dechambre appear to conceive that there is no intermediate state between their congestive erection of the cells, and suppuration.

There is a condensation of the lung, destitute of the granular appearance, which is considered by Andral and some others, due to the agency of inflammation, as well as the granulated condensation. Speaking of pneumonic condensation, he says, after describing the granulated appearance of some lungs, "In other cases, the granular appearance is altogether wanting, and its surface, when cut, appears perfectly smooth,"—p. 510.[2] This he refers to the greater degree of the tumefaction of the air cells; "for when the tumefaction passes a certain limit, its effect is to approximate the cells so closely, that they become confounded together, and the granulated appearance vanishes entirely." Williams, Hourmann, and Dechambre, take a different view of the matter, and consider the smooth condensation (*engorgement non-granulée*), as the result of an affection of a different set of vessels from those engaged in the granular condensation. They therefore consider it a separate form of pneumonia, and term it the "intervesicular."[3] It is, however, far from being proved, that this condensation is entitled to be ranked among the effects of inflammation. It is described correctly as of a dark colour and smooth section, yielding more or less of a bloody serum when squeezed, destitute of crepitation, and often sinking in water to the bottom of the vessel. It is in fact the sort of condensation which sometimes occurs in our continued fevers, and is common in such cases of disease, of whatever kind, as present an obstacle to the easy transmission of blood through the lungs. There is a strong presumption in favour of the opinion, that it is the result of passive congestion merely, in the fact of its being the most remarkable at the depending parts of the lungs. Hourmann and Dechambre, although they conceive it to be of inflammatory origin, and designate it the intervesicular variety of pneumonia, allow that there is a presumption in favour of its being of the nature of engorgement; and in summing up the grounds on which a non-granular engorgement may be deemed inflammatory, they say,—"The inflammatory nature of the congestion may be concluded always, if it occupy either the anterior border or the whole extent of the organ, when at the same time, there does not exist in the heart or in the large vessels any obstacle to the circulation. But we must allow, that the first degree of engorgement is the only one which we have observed in such circumstances, and with such conditions."[4] In other words, they have never seen the smooth condensation, except at the depending parts.

[1] Cycl. of Pract. Med. p. 410.
[2] Path. Anat. Translation.
[3] Librar. of Med., vol. iii.
[4] Arch. Gén., t. x.

Professor John Reid of St Andrews, having instituted some experiments in order to ascertain the effect of division of the par vagum on the lungs, found that in fifteen out of seventeen instances, a morbid state of these organs resulted. In eight cases, there was more or less of the planiform condensation, and in five, either granular hepatisation or infiltration with pus. If the state of the lungs in these thirteen cases is to be considered as the result of inflammation, we have the proportion of the intervesicular to the vesicular pneumonia, very remarkably inverted by the division of the eighth pair of nerves; for Hourmann and Dechambre found in eighty-eight cases which they designate pneumonia, the smooth condensation in only eighteen, or nearly in the proportion of only one to four of the granulated condensation. We know nothing to countenance a supposition that the experiments of Dr Reid can incline to the inflammation of one tissue of the pulmonary parenchyma in preference to another; but there can be no question that the effect of these experiments, —the diminution of the respiratory muscular movements—has the power of interrupting the freedom of the circulation through the lungs, that is, of producing congestion there. That actual pneumonia should ensue in some cases, on extreme congestion, is no more than might be anticipated.

The third case to which I referred in an early part of this paper, as having exhibited a degree of engorgement so great, that the dense tissue, when cut into small portions, sank readily in water, afforded a specimen of this kind of condensation. By examining it, I was enabled to determine, that neither the globule of inflammation, such as I have described it, nor altered epithelium cells, existed in it. The latter, on the contrary were, as I have said, quite normal in their appearance. And more than this, it was made very plain by the kind of fluid which could be expressed from the smaller bronchial tubes, that the congestion did not exist solely, even in its greatest degree, in the intervesicular tissue, for the fluid in question contained not only the colouring matter of the blood, but the entire blood-discs themselves in great abundance.[1] This latter error of the French pathologists, to whom I have referred, was doubtless the offspring of the former. For if pneumonic condensation, in its red state, must consist of sanguineous engorgement only, and one kind of it be supposed to exist in the blood-vessels of the cells,—the other kind has no place left for it but the intervesicular tissue.

But although satisfied that the intervesicular pneumonia, as described by the authors who have introduced the name, is nothing else than extreme sanguineous engorgement of the tissue

[1] Professor Reid had conjectured that such was probably the case in the condensed lungs produced in his experiments. Edin. Med. and Surg. Journal, No. 139.

of the lungs of the passive kind, I am far from doubting the occasional occurrence of inflammation, of such intensity, in the common cellular tissue, as to produce the principal morbid appearance in that situation. A case in which this actually occurred is recorded in my paper on the Pathology of Laryngismus, in the first number of this Journal. It is there stated, that the purulent infiltration was principally in the interlobular cellular tissue, and that some of the interlobular spaces were considerably widened, in consequence of their containing much purulent matter. In that case, the inflammation had extended from the cellular tissue of the neck and mediastinum, through the pleura to the cellular tissue of the lungs; and it was remarkable that the condensation of the lung took place without the occurrence of the crepitant rattle. Cases of this kind, however, are very rare; and as yet nothing is known of the appearance which the lungs present, prior to the occurrence of purulent infiltration, when the inflammation exists, either solely or chiefly, exterior to the mucous membrane.

In treating of the granulations of pneumonia, I have noticed them as they occur in connection with a viscid or thickish state of the secretion of the air cells. They do not, however, always occur in connection with this. I have seen a granulated surface produced by the accumulation of a fluid, as thin as that of the first stage of pneumonia. The granulations in this case are not large, and persist but a short time after the section is made which exposes them, because the thin fluid which occasions them readily escapes. The colour which accompanies this thin fluid may be either a very pale red, or it may be of the deepest sanguineous tint. When the latter is the case, as it usually is at the lower parts of the lungs, the appearance termed splenisation is presented. There is then an imperfect and transitory appearance of granulation, and a very wet surface, in addition to the dark colour. This state is to be found occasionally, and sometimes with patches interspersed of more perfect granulation, as in the hepatised lung, in the depending portions of the lungs of persons who have died after a prolonged agony, when it is termed the *pneumonia agonisantium*, and in those who have suffered for a time from an impediment to the free transmission of blood through the lungs, from whatever cause. Laennec seems to have taken this appearance for a certain proof of inflammation having existed; for it is to it he alludes in the following passage:—"The sero-sanguineous congestion of the lungs, which takes place in almost all dying persons, is frequently converted into pneumonia, if the agony is at all protracted. On examination after death, different points of the lungs are found distinctly hepatised."[1] I

[1] On Diseases of the Chest, p. 341.

do not know how the true nature of this appearance can be determined, without an examination of the infiltrated fluid by means of the microscope. In some instances of it I have been able to trace satisfactorily the evidences of inflammation, by the globules of inflammation and the altered epithelium cells; while, in others, perfectly alike in their more familiar characters, the microscopic tests of inflammation have been absent. Granulation, in these latter cases, must have been the result of distention of the cells by passive exudation, which I have not seen produce this effect, except at the depending parts of the lungs, where, of course, the pressure of the accumulated fluid would produce the most effect on the air cells.

Explanation of the Plate.

Figure 1. Globules of inflammation entire, and broken down. Magnified about 500 diameters.

Figure 2. Epithelium cells from the air cells of passively engorged lungs. Magnified about 500 diameters.

Figure 3. Epithelium cells as altered by inflammation, from the air cells of pneumonic granulations. Magnified about 500 diameters.

Figure 4. Pus globules as contained in the same situation. Magnified about 500 diameters.